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Control of Refractory Ventricular Ectopy

Atrial and Ventricular Overdrive Pacing

ROY MEYER, MD
LESTER B. JACOBSON, MD
San Francisco

THE SUPPRESSION of ventricular arrhythmias by cardiac pacing was first reported by Zoll and coworkers,[1] who used transcutaneous thoracic ventricular pacing to prevent the onset of ventricular tachycardia (VT) and ventricular fibrillation (VF) in patients with *complete* atrioventricular (AV) block and *slow* ventricular rhythms. Four years later Sowton and colleagues[2] reported the successful suppression of VT and VF in patients with *normal* AV conduction and *normal* ventricular rates by right ventricular endocardial pacing at rates exceeding the spontaneous sinus rate. During the past 15 years it has been clearly shown that ventricular[3-6] and atrial[6-9] pacing may be valuable techniques for suppressing otherwise intractable ventricular arrythmias that occur in circumstances involving myocardial ischemia,[3] cardiac operation[3,4] and medications that produce arrhythmias,[3,5] as well as in the absence of underlying heart disease.[7]

ABBREVIATIONS USED IN TEXT
AV = atrioventricular
LV = left ventricular
PAW = pulmonary artery wedge (pressure)
PVC = premature ventricular complex
VA = ventriculoatrial
VF = ventricular fibrillation
VT = ventricular tachycardia

Despite a large body of information on the hemodynamic effects of cardiac pacing in *stable* patients with and without heart disease, the hemodynamic effects of cardiac pacing in *unstable* patients with serious ventricular arrhythmias have not been adequately described, most likely because most reports on overdrive suppression were published before the availability of simple techniques for bedside hemodynamic monitoring. This case report illustrates the use of temporary atrial and ventricular pacing for overdrive suppression of serious ventricular arrhythmias in a patient several weeks after acute myocardial infarction. It documents and compares the changes in cardiac output and pulmonary artery wedge (PAW) pressure that occurred with suppression of the arrhythmia by atrial and ventricular pacing, as well as by antiarrhythmic medication.

Report of a Case

A 54-year-old man had an acute myocardial infarction during which pathological Q waves appeared in leads V_{1-4} and serum creatine kinase levels reached a peak of 3,252 IU per ml (normal 50 to 180 IU per ml). His initial hospital course was complicated by premature ventricular complexes (PVC's), which resolved following intravenous administration of lidocaine, and hypotension, which resolved with intravenous administration of modest amounts of fluids. After 15 days in hospital the patient was discharged with instructions to take propranolol, 20 mg four times a day, because he was having intermittent chest discomfort that suggested myocardial ischemia. In the ensuing two weeks severe shortness of breath developed to the point where he had orthopnea, and he began to be aware of abnormal cardiac activity. He was readmitted to hospital and found to be in severe congestive heart failure and to have

From the Division of Cardiology, Presbyterian Hospital of Pacific Medical Center, San Francisco. Dr. Jacobson is now at the San Francisco Kaiser Foundation Hospital.

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Reprint requests to: Roy Meyer, MD, Division of Cardiology, Presbyterian Hospital, P.O. Box 7999, San Francisco, CA 94120.

frequent multiform PVC's, occasionally as many as four in a row. Initial treatment with sequential intravenous administration of large doses of lidocaine and procainamide did not alter the arrhythmia, so 20 mg of propranolol every six hours and 200 mg of disopyramide (Norpace) every eight hours were administered orally. However, the additional medication did not suppress the arrhythmia. Within 36 hours of starting the propranolol and disopyramide therapy, the congestive heart failure state became more profound, and a Swan-Ganz thermodilution catheter was inserted. The mean pulmonary artery wedge pressure measured 28 mm of mercury, so diuretics were administered intravenously. The patient was transferred to Pacific Medical Center for evaluation and treatment.

On admission, the patient looked and felt very weak. He appeared to have moderately low cardiac output with moderate jugular venous distention and diffuse rales that suggested high intracardiac filling pressures. There was a large area of anterior wall dyssynergy and a prominent S_3 gallop. An electrocardiogram (ECG) (Figure 1) showed abnormalities compatible with the recent anterior wall myocardial infarction and multiform PVC's singly, in pairs and in short runs. About 60 percent of the QRS complexes were PVC's. The initial mean PAW pressure was 19 mm of mercury, and cardiac output determined in triplicate by the thermodilution technique was 2.8 liters per minute (cardiac index = 1.5 L/min/m^2). Laboratory values for serum electrolytes, creatinine, creatine kinase and blood urea nitrogen were within normal limits. The liver function tests showed mild abnormalities compatible with hepatic congestion.

Because the PVC's had persisted despite the addition of large doses of intravenously given lidocaine, the decision was made to attempt to suppress them with pacing techniques. Therefore, a 4F bipolar electrode catheter was introduced percutaneously into a left antecubital vein and, using electrocardiographic monitoring (with the pin from the tip electrode attached to the V1 terminal of the ECG machine) advanced to an area in which the atrial electrogram was very prominent (Figure 2). Consistent atrial capture from this area was achieved with a current output of about 2 mamp using the Medtronic Model 5880A external pacemaker generator. As shown in Figure 3A, asynchronous atrial pacing was initiated at a rate of about 105 per minute, and the rate was gradually increased until ventricular ectopic activity was consistently suppressed, which occurred at an atrial rate of about 125 beats per minute. However, as shown in Figure 3B, at rates slightly below 120 beats per minute ventricular ectopic activity reappeared. After 30 minutes of atrial pacing at a rate of about 125 beats per minute, there was a dramatic increase in cardiac output from 2.9 to 4.8 liters per minute and a fall in the mean PAW pressure from 19 to 14 mm of mercury. Figure 4 displays a plot of cardiac output against mean PAW pressure to display the serial hemodynamic changes that occurred as a result of changes in cardiac rhythm.

In an attempt to suppress the PVC's with antiarrhythmic medications, quinidine sulfate and phenytoin (Dilantin) were administered orally

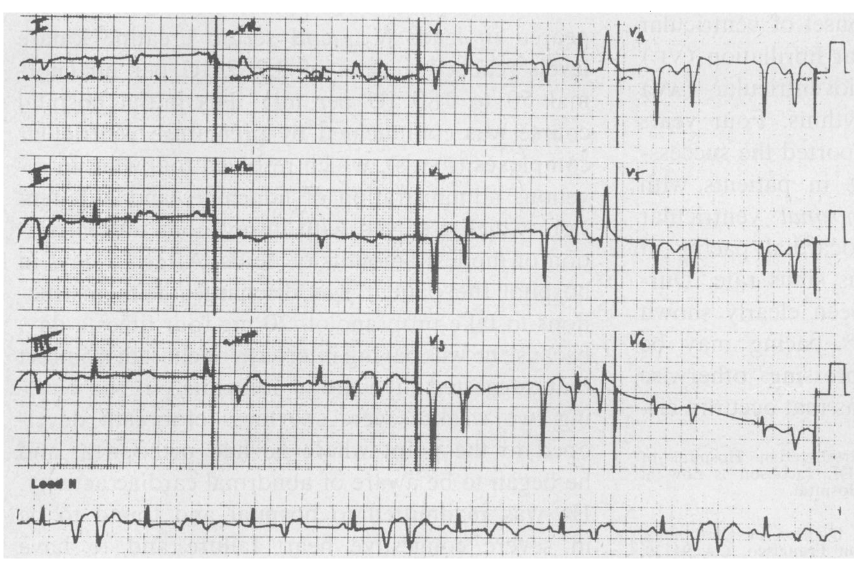

Figure 1.—Twelve-lead electrocardiogram showing PVC's and abnormalities compatible with the recent anterior wall myocardial infarction. The lead II rhythm strip displays the frequency and group beating pattern of PVC's (PVC's = premature ventricular complexes).

while atrial pacing was continued at a rate of about 125 beats per minute. Within hours after these medications had been given, the initially suppressed PVC's reappeared in a bigeminal pattern, as their coupling intervals became progressively shorter than the paced atrial cycle length.

Although PVC's could be completely suppressed at this time by increasing the atrial pacing rate to slightly more than 130 beats per minute, the pacing rate was kept at about 125 beats per minute and the bigeminal rhythm allowed to occur. Serial measurements during atrial pacing indicated that the cardiac output was lower in the presence of the PVC's than in their absence and, at identical PAW pressures of 10 mm of mercury, the cardiac output was higher when the PVC followed a paced P wave than when it occurred almost simultaneously with it (4.5 versus 4.1 liters per minute).

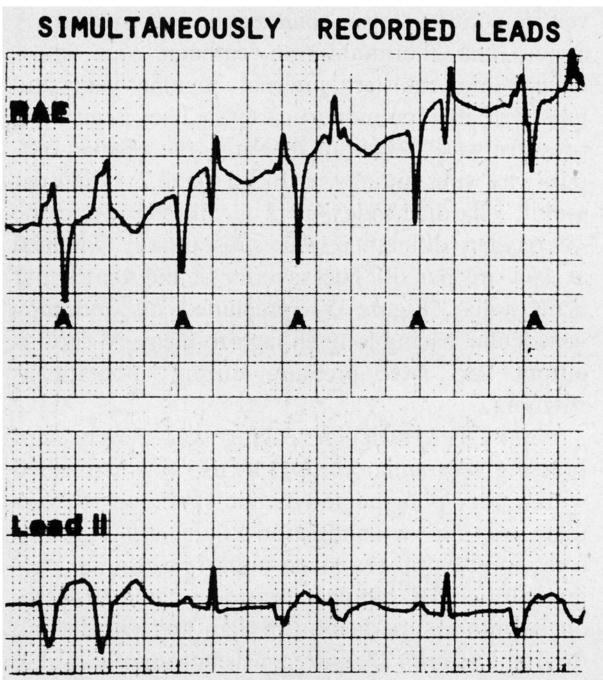

Figure 2.—Simultaneously recorded unipolar right atrial electrogram (RAE) and lead II. The RAE is recorded in the area from which atrial pacing was carried out (A = atrial deflections).

To see the effect of the antiarrhythmic medication alone on the spontaneous rhythm, pacing was stopped briefly every two hours. Atrial pacing was discontinued after 28 hours when stable sinus rhythm with only rare PVC's were present. An hour after cessation of atrial pacing, the cardiac output was 3.9 liters per minute and the mean PAW pressure was 12 mm of mercury. The dramatic rise in cardiac output and the fall in mean PAW pressure between the initial and final points in Figure 5 reflect solely the beneficial effects of suppression of the PVC's.

Over several days the patient increased his activities and felt reasonably well. Cardiac catheterization, left ventricular cineangiography and selective coronary arteriography were done to assess the advisability of operative intervention. The findings showed significant left ventricular

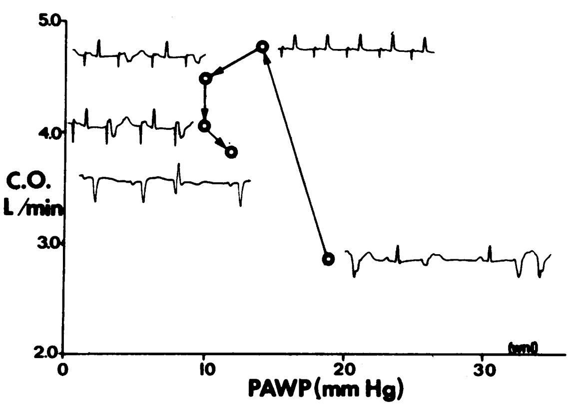

Figure 4.—Display of serial changes in cardiac output (C.O.) and mean pulmonary artery wedge pressure (PAWP) with changes in rhythm during therapy. The increase in C.O. and the fall in mean PAWP between the initial and final points reflect the beneficial effects of suppression of PVC's by antiarrhythmic medications (PVC's = premature ventricular complexes).

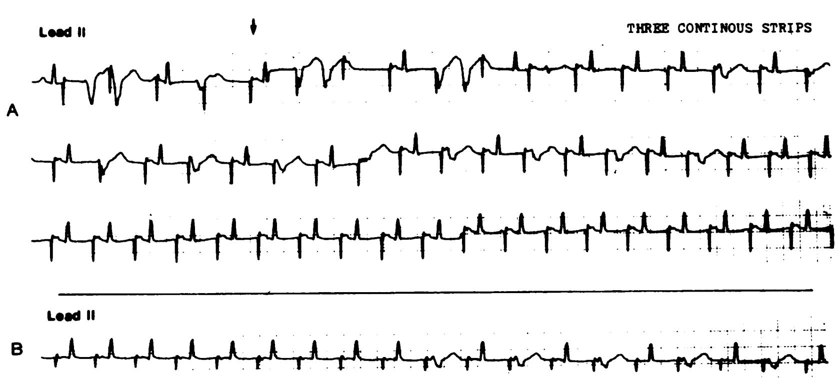

Figure 3.—**A,** Three continuous lead II rhythm strips recorded as atrial pacing is initiated and the pacing rate increased. Consistent atrial capture begins at the arrow. Complete suppression of PVC's occurs at a paced atrial rate of about 125 beats per minute. **B,** As the atrial pacing is slowed from 125 to 118 beats per minute the PVC's reappear (PVC's = premature ventricular complexes).

(LV) dysfunction with a very large LV end-diastolic volume, large anterior, apical and septal dyskinetic segments, a mildly elevated LV end-diastolic pressure of 14 mm of mercury, and a severely reduced calculated ejection fraction of 0.36. The cardiac output, determined by the dye dilution technique, was 4.4 liters per minute and the aorta-right atrial oxygen difference was 4.9 ml per dl when the rhythm was sinus at a rate of 90 per minute with rare PVC's. Selective coronary arteriography showed total occlusion of the proximal portion of the left anterior descending coronary artery, but in the remaining branches of the left coronary artery system and the entire right coronary artery system no abnormalities were noted. It was felt that LV aneurysmectomy was not indicated because the arrhythmia was adequately controlled with medication, and inadvisable because the interventricular septum, which appears to play an important role in postmyocardial infarction ventricular arrhythmias,[10] could not have been resected. The patient was discharged with instructions to take 1,000 mg of quinidine per day (which had resulted in a plasma quinidine level of 3.2 mg per liter) and 300 mg of phenytoin per day.

At home the patient resumed his usual sedentary activities and did well for about five weeks until he suffered an unheralded cardiac arrest. He was resuscitated by paramedics who found him to be in ventricular tachycardia, which they terminated with direct current countershock. Following cardioversion the rhythm was sinus with frequent PVC's which could not be suppressed despite intravenous administration of lidocaine, procainamide, propranolol and phenytoin. So, using a temporary bipolar electrode catheter introduced into the right internal jugular vein and located with its tip probably in the middle coronary vein, demand ventricular pacing was initiated at a rate of about 80 beats per minute; the rate gradually increased to about 110 per minute, at which time suppression of almost all PVC's was achieved. At this point the patient was transferred back to Pacific Medical Center. There had been no evidence of acute myocardial necrosis at the time of the cardiac arrest.

On readmission the patient was weak and seemed to have moderately low cardiac output with high intracardiac filling pressures. The systemic pressure was variable but averaged about 95/60 mm of mercury during a paced ventricular rhythm at a rate of about 110 beats per minute. During ventricular pacing there was AV dissociation with occasional sinus capture beats because the sinus rate was about 92 per minute and no ventriculoatrial (VA) conduction was present. A Swan-Ganz thermodilution catheter was introduced into the left internal jugular vein and passed to the pulmonary artery. The mean PAW pressure was 21 mm of mercury, the cardiac output was substantially reduced at 2.2 liters per minute (cardiac index = 1.2 L/min/m^2), and the AV oxygen difference was substantially widened at 10.7 ml per dl. The severity of the ventricular arrhythmia (Figure 5) precluded discontinuing ventricular pacing long enough to measure cardiac output and PAW pressure during spontaneous rhythm.

Although ventricular pacing was clearly suppressing ventricular ectopy, its use was associated with a very poor hemodynamic state even though there was no VA conduction at this time. Therefore, an attempt was made to carry out atrial pacing with a 4F bipolar electrode catheter introduced into a superficial arm vein and passed into the right atrium. Despite consistent atrial capture, complete suppression of the PVC's did not occur at rates as high as 145 beats per minute because of the short coupling intervals of the PVC's. Thus,

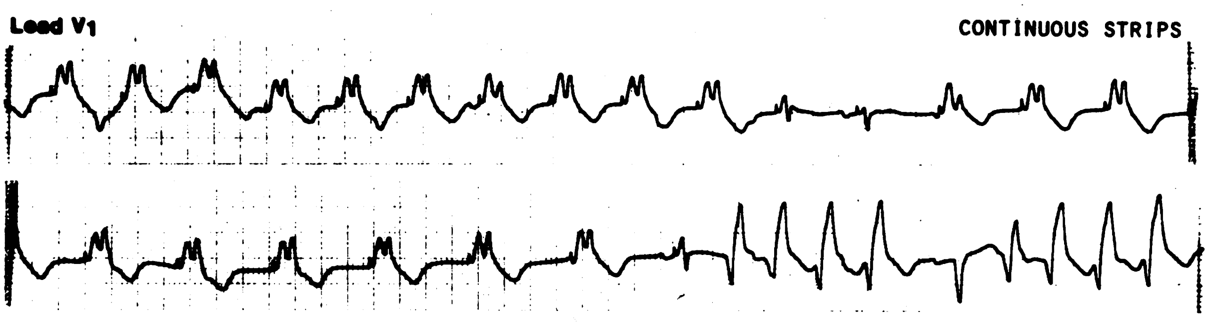

Figure 5.—Two continuous lead V_1 rhythm strips recorded as the rate of ventricular pacing is decreased from about 113 beats per minute. When the paced ventricular rate reaches about 76 beats per minute salvos of PVC's appear (PVC's = premature ventricular complexes).

ventricular pacing at 110 per minute was continued while 300 mg per day of phenytoin was given. The dosage of quinidine sulfate was increased from 1,000 to 2,400 mg per day to achieve higher therapeutic plasma levels. During ventricular pacing the cardiac output ranged from 2.2 to 2.9 liters per minute and the mean PAW pressure stabilized at 17 to 18 mm of mercury. Ventricular pacing was continued for 14 days, until spontaneous PVC's did not occur during sinus rhythm. In the ten days following discontinuation of ventricular pacing, the patient was able to walk about the hospital with only rare PVC's, without angina pectoris and with adequate control of congestive heart failure. He was discharged with instructions to take 300 mg per day of phenytoin and 2,400 mg per day of quinidine sulfate. Over eight months he has continued do do well.

Discussion

This case illustrates the value of cardiac pacing as a temporizing measure in the control of ventricular ectopic activity, and shows that pacing the atrium or the ventricle may be equally effective in this regard. Furthermore, it shows the considerable hemodynamic improvement that may occur with atrial overdrive pacing and documents the superiority of atrial over ventricular pacing in optimizing the hemodynamic state in a patient with LV dysfunction. The dramatic rise in cardiac output and the fall in mean PAW pressure that occurred with atrial overdrive suppression in this patient are not attributable to an increase in heart rate, for the average ventricular rate before atrial pacing was about 120 beats per minute (Figure 1), similar to the ventricular rate during atrial pacing. Rather, this dramatic hemodynamic improvement reflects (1) the suppression of ventricular ectopic beats which occur at such short coupling intervals that the abbreviated diastolic filling time results in low end-diastolic volume and, thus, small stroke volume on the basis of the Frank-Starling mechanism, and (2) preservation of the normal atrial and ventricular contraction sequences which, in the setting of LV dysfunction, may have a profoundly beneficial effect on LV stroke volume.[11] The fact that during ventricular bigeminy at identical PAW pressures of 10 mm of mercury the cardiac output was higher when the PVC followed a paced P wave than when it occurred almost simultaneously with it indicates that the coupling interval of a PVC may be of hemodynamic significance by virtue of its relationship to atrial contraction as well as its absolute degree of prematurity.

In this patient the beneficial effects of a properly timed atrial contraction on cardiac output are demonstrated by the substantially higher cardiac output (4.8 versus 2.2 to 2.9 liters per minute) during atrial pacing than during ventricular pacing at comparable rates and at comparable LV filling (mean PAW) pressures. While the rapid ventricular rates required to reduce ventricular ectopic activity might be expected to reduce cardiac output, it is clear from this and other reports[12] that cardiac output may not fall at high pacing rates and that at any given pacing rate the hemodynamic changes will depend on the chamber (atrium versus ventricle) paced,[12] the degree of suppression of ventricular ectopic activity achieved and the coupling intervals of the PVC's to the spontaneous QRS complexes. Furthermore, growing evidence suggests that the theoretical risk of inducing myocardial ischemia with rapid pacing has been overestimated because the rapid pacing rates have generally been well tolerated by patients with coronary artery disease and myocardial ischemia.[3,13] Often, the addition of antiarrhythmic medications allows the pacing rate required for suppression of PVC's to be lowered, even if such drugs were not initially effective in completely suppressing the arrhythmias.[6] Similarly, an arrhythmia resistant to either pacing at an acceptable rate or to antiarrhythmic medication alone may respond to the simultaneous use of both therapeutic modalities.[7]

Atrial pacing is the preferred mode of suppressing ventricular ectopic activity because it provides the hemodynamic advantage of atrial and ventricular synchrony which, in turn, allows rapid ventricular rates to be achieved without hemodynamic deterioration. However, when its use is precluded by the presence of atrial standstill, flutter and fibrillation, ventricular pacing may be effectively employed. In the presence of sinus rhythm with high grade AV block, AV sequential pacing would be expected to be the preferred modality[14] and might, during our patient's second hospital stay, have provided hemodynamic improvement in addition to PVC suppression.

Achieving consistent atrial capture at low-energy output for longer than several hours is often not possible when pacing is carried out, as was the case in our patient, with the electrode catheter tip floating freely within the right atrium.

Therefore, when it is anticipated that atrial pacing will be needed for prolonged periods of time, it is preferable to position fluroscopically specially designed electrode catheters into the coronary sinus or right atrial appendage.

It is unclear in our patient's case whether improvement in the hemodynamic state per se played a role in the suppression of the PVC's; however, the fact that PVC's were completely abolished with ventricular pacing at a rate of 110 beats per minute at a time when the cardiac output was very low and the mean PAW pressure was 17 to 18 mm of mercury, suggests that hemodynamic improvement is *not necessary* for the effective suppression of ventricular ectopic activity by overdrive pacing. This observation and the conclusion of a recent review article[13] suggest that the phenomenon of overdrive pacing may be explained on a purely electrophysiological basis. However, as the exact mechanisms responsible for ventricular ectopic activity in humans are still uncertain, the mechanisms by which rapid pacing suppresses ventricular arrhythmias remain unclear. Early workers, believing that ventricular ectopic activity reflected enhanced automaticity, hypothesized that rapid pacing evoked changes in electrolyte and autonomic milieu, events that depress automaticity by increasing the stimulation threshold and by reducing the rate of spontaneous depolarization and the amplitude of the action potential of an ectopic focus.[13,15] More recent reports, advocating reentry as the mechanism of ventricular ectopic activity, suggest that rapid pacing suppresses ventricular ectopic activity by causing changes in conduction velocities and in refractory periods of tissues making up the reentrant pathways.[16,17] However, as many patients successfully treated with rapid pacing have had long QTU intervals before pacing,[4,5,8,9,18-20] it is possible that the phenomenon of overdrive suppression involves rate-related shortening of the Purkinje fiber action potential.

Conclusions

Overdrive pacing for the suppression of ventricular arrhythmias may be an effective temporizing measure employed to allow time for adequate levels of antiarrhythmic medications in the blood and tissues to be achieved or for an acute process such as drug effect, myocardial ischemia or infarction to resolve. However, for patients with recurrent life-threatening ventricular arrhythmias, long-term overdrive suppression may be achieved with permanent atrial and ventricular pacing systems.[2,6-8,18] Although this technique has not received much attention in recent literature, it deserves reemphasis as a valuable therapeutic modality which may have dramatic antiarrhythmic and beneficial hemodynamic effects in specific clinical situations.

Summary

Temporary atrial and ventricular pacing was carried out to suppress refractory ventricular arrhythmias in a patient in whom acute myocardial infarction had occurred several weeks earlier. Although pacing from both locations was effective in suppressing ventricular arrhythmias, atrial pacing provided greater hemodynamic improvement.

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